Effect of transpulmonary and driving pressures on collateral gas flow in dog lungs

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OLSON, L. E. *Effect of transpulmonary and driving pressures on collateral gas flow in dog lungs.* J. Appl. Physiol. 59(6): 1757–1765, 1985.—The effect of changing segment pressure (Ps) and airway opening pressure (Pao) on flow through a collaterally ventilating lung segment was evaluated in intact and excised dog lungs. He, N_2, Ne, and SF_6 were passed through the lung segment distal to a catheter wedged in a peripheral airway at driving pressures (Ps − Pao) between 0.25 and 2 cmH_2O. Eight excised caudal lobes were studied at Pao = 5, 10, and 15 cmH_2O. Flow was directly related to Ps − Pao and Pao and inversely related to the density of the gas. A dimensionless plot of the driving pressure normalized to a reference dynamic pressure as a function of Reynolds number (Re) indicated that flow through the segment behaved as if it were laminar at Re <100 and that increasing Pao increased the dimension of the pathways conducting flow as shown previously. Small changes in Ps had no effect on pathway geometry or on the pattern of flow through the segment at Pao = 10 and 15 cmH_2O. At Pao = 5 cmH_2O increasing segment pressure appeared to increase the dimensions of the flow pathways slightly. Similar changes in Ps − Pao had no consistent effect on flow pattern or pathway geometry in six anesthetized, paralyzed, vagotomized dogs at functional residual capacity or after widely opening the chest (Pao = 5 cmH_2O). These results suggest that, at large lobe volumes, airways (including collateral pathways) are maximally dilated and therefore relatively insensitive to small changes in segment pressure. In excised lobes at low volumes, where both airways and parenchyma are more distensible, raising segment pressure appears to increase the dimensions of the pathways conducting flow through the collaterally ventilating lung segment.

intact lungs; excised lungs; pressure-flow relationship; Moody diagram

COLLATERAL VENTILATION is important in maintaining ventilation distribution in lungs in which time constant inequalities cause nonuniformities in regional lung volume (6, 15). Although it is unclear whether interalveolar pores or small airways are the primary route for collateral ventilation, studies examining the influence of lung volume (8, 10, 18), CO_2 (21, 25), and parasympathetic tone (17, 22) on collateral gas flow suggest that the structures behave like airways.

The resistance to collateral gas flow is quantified by measuring the pressure necessary to cause a known flow through a collaterally ventilating lung segment (5). Because the segment is distensible and continuous with the surrounding parenchyma, it is likely that measuring collateral resistance causes a change in regional lung volume. Collateral resistance might therefore be expected to be a function of the pressure used to measure it. Yet studies examining the effect of alterations in driving pressure on resistance are contradictory. Increasing segment pressure has been reported to increase collateral resistance in excised horse lungs and increase or decrease collateral resistance in excised and intact dog lungs (4, 10, 19). The observation that resistance may vary with driving pressure indicates that the collateral pressure-flow ($P\dot{V}$) relationship is not a simple straight line. Nonlinearities in the $P\dot{V}$ relationship can be caused either by a pressure-dependent change in the geometry of the flow conduits or a change from viscosity to density-dependent airflow.

The relationship between driving pressure and resistance should depend on the degree of mechanical interaction, or interdependence (12), between the segment and its surroundings and the deformability of the parenchyma and intrasegmental airways/collateral pathways. Studies quantifying interdependence by measuring the effective compliance of obstructed lung regions indicate mechanical interactions between adjacent lung regions to be greater in unlobulated dog lungs than lobulated pig and calf lungs and interdependence between a lung region and the chest wall to be greater than between adjacent lung regions (13, 14, 20, 24, 26).

The present study was designed to evaluate the effect of small alterations in driving pressure on the collateral $P\dot{V}$ relationship. $P\dot{V}$ measurements were made at three transpulmonary pressures in excised dog lungs to evaluate the effect of altering parenchymal stiffness. Measurements were also made in intact dog lungs before and after widely opening the chest to determine whether the presence of the chest wall altered the $P\dot{V}$ relationship. Dimensional analysis was used to evaluate the functional relationship between driving pressure and collateral flow to determine whether nonlinearities were due to a change from viscosity to density-dependent flow or a change in the geometry of the pathways conducting flow through the collaterally ventilating lung segment.

METHODS

Excised Lungs

Mongrel dogs of both sexes were anesthetized with pentobarbital sodium (20 mg/kg iv), heparinized (1,000

U), and exsanguinated. The heart and lungs were excised en bloc and the caudal lobes carefully removed. The lobar vessels were ligated and a Y connector tied into the lobar bronchus. The lobes were weighed and vacuum degassed, and a pressure-volume (PV) curve was recorded following several large inflations. Total lobe capacity was defined as lobe volume when pressure at the lobar bronchus was 25 cmH_2O. Minimum volume was determined by water displacement. Some lobes were refrigerated overnight to reduce gas trapping. Because results appeared identical between refrigerated and nonrefrigerated lobes the data were pooled. A double-lumen catheter (0.5 cm OD) was advanced through one arm of the connector into the inflated lobe until it became securely wedged in a subsegmental bronchus. The outer lumen was used to deliver humidified He, N_2, Ne, or SF_6 to the lung segment distal to the wedged catheter, and the inner lumen was used to monitor pressure in the segment (Ps). The gases were chosen to provide a wide range of densities and viscosities (see Table 1). Gas flow through the catheter, and therefore through the collaterally ventilating lung segment, was determined with a pneumotachograph (Fleisch no. 0000) calibrated by timed collections of each gas. Flow ($\dot{V}$) and the pressure driving flow, the difference between Ps and pressure at the lobar airway opening (Pao), were recorded on a polygraph (Gould model 2400S) using Validyne transducers (model DP 45) calibrated daily against a water manometer. The lobe was inflated through the second arm of the lobar connector, and Pao was monitored with a water manometer. Because the lobes were excised, Pao equaled transpulmonary pressure (Ptp). The lobes were covered with wet gauze and occasionally sprayed with saline to prevent drying.

After a large inflation, Pao was adjusted to 15, 10, or 5 cmH_2O, and flow through the wedged catheter increased until Ps − Pao = 2 cmH_2O. Flow recorded as Ps − Pao was decreased from 2 to 0 cmH_2O in 0.25 cmH_2O increments. The lung segment was then flushed with the next test gas, and the sequence was repeated for each Ptp.

Data analysis. Flow through the collaterally ventilating lung segment was recorded as a function of Ptp and driving pressure for four different gases. A repeated-measures design three-way analysis of variance (ANOVA) was used to determine the significance of these effects and their interactions. Prior to analysis, a log transformation was applied to the flow data to assure homogeneity of variance. Significance was determined at the $P < 0.05$ level.

Although ANOVA confirmed that flow was a function of Ptp, driving pressure, and the physical properties of the test gas, the critical question of how these factors interact remained. Dimensional analysis was used to answer this question.

TABLE 1. *Gas physical properties*

	He	Ne	N_2	SF_6
Viscosity (μ), poise	1.96×10^{-3}	3.18×10^{-3}	1.74×10^{-3}	1.53×10^{-3}
Density (ρ), g/l	0.166	0.836	1.16	6.14

From Engineering Report 131, Matheson Inc., and *Handbook of Chemistry and Physics* (48th ed.), Cleveland, OH: CRC.

The application of dimensional analysis to pulmonary physiology is well documented (1, 9, 18, 23). Briefly, driving pressure, average flow, and gas density (ρ) and viscosity (μ) were combined into two nondimensional parameters, driving pressure normalized to kinetic pressure (P_n) and Reynolds number (Re).

P_n was computed as

$$P_n = \frac{2(Ps - Pao)A^2}{\rho \dot{V}^2}$$

This parameter is similar to the coefficient of friction but uses static pressure losses (Ps − Pao) in the numerator rather than total frictional losses [(Ps − Pao) + $\frac{1}{2}\rho\dot{V}^2/A^2$] as kinetic losses were negligible. Reynolds number was computed as

$$Re = \frac{\rho d \dot{V}}{\mu A}$$

Area (A) was calculated for each Ptp using the diameter (d) of the circular catheter tip. A double-log plot of P_n against Re, conventionally referred to as a "Moody-type" plot, was then constructed.

The advantage of this analysis is that it distinguishes changes in flow regime from changes in the dimension of the flow conduits. The flow regime is indicated by the functional relationship between the computed variables. When log P_n and log Re are inversely related with a slope of −1, flow is viscosity dependent and the resulting equation simplifies to the familiar Hagan-Poiseuille relationship. When log P_n is independent of log Re, i.e., the slope is zero, flow is density dependent and the resulting equation shows Ps − Pao to be proportional to $\dot{V}^2$. Transitional flow is indicated by a curve having a slope between these two extremes. The position of the curve indicates whether the geometry of the flow conduits was constant or variable as pressure and flow were altered as discussed below. The dimension of the catheter tip was used as the characteristic dimension (d) because that was where pressure and flow were known. Although many Re values exist in the branching network of flow conduits within the segment, all existing Re values are proportional to the computed Re and therefore inferences from the Moody plot hold for flow through the entire segment.

Intact Lungs

Mongrel dogs of both sexes were anesthetized with pentobarbital sodium and atropinized to reduce pulmonary secretions. Four of the dogs were intubated, and a balloon-tipped catheter was placed in the distal third of the esophagus. Ptp was measured as the difference between tracheal pressure and esophageal pressure. After several large inflations, the lungs were inflated to a Ptp of 25 cmH_2O and Ptp was recorded as lung volume was reduced to residual volume in a stepwise manner. PV curves appeared normal in each dog.

All dogs were studied in the supine position. A three-way connector was tied into the trachea through which

a bronchoscope could be passed while ventilation was maintained with a fixed-volume ventilator. Succinylcholine chloride was given as necessary to suppress spontaneous respirations, and the vagi were cut to reduce reflex changes in airway tone. Some dogs breathed an enriched O_2 mixture. A bronchoscope (tip diam = 0.5 cm) was wedged in a caudal lobe, and test gases were delivered to the distal lung segment through the suction port. A small catheter in the suction port measured pressure at the bronchoscope tip, which was used to estimate pressure in the distal lung segment. Flow through the bronchoscope was measured as described previously, and $\dot{V}$, Ps − Pao, and Ptp were recorded on a polygraph (Hewlett-Packard) and *X-Y-Y* recorder (Hewlett-Packard model 7046B) using Validyne transducers. After flushing the collaterally ventilating lung segment with the test gas, the ventilator was switched off and the lungs were allowed to deflate passively to functional residual capacity (FRC). Driving pressure was increased to ~2 cmH_2O by raising segment pressure and then decreased in unselected intervals while flow was recorded. The lungs were sighed frequently to minimize atelectasis. The chest was then widely opened by a lateral thoracotomy, lung volume was maintained by applying 5 cmH_2O positive pressure at the trachea, and the sequence was repeated.

Data analysis. Data from intact lobes were not averaged or analyzed statistically because $\dot{V}$ was not measured at identical driving pressures. Normalized pressure and Re were therefore computed and plotted for each individual dog.

RESULTS

Excised lobes

The ANOVA indicated that flow was a strong function of Ptp, Ps − Pao, and the physical properties of the test gas. Sample data from a single lobe are shown in Fig. 1. Because these are actual records, and $\dot{V}$ was measured with a pneumotachograph, $\dot{V}$ is represented in pressure units. Figure 1*A* shows $\dot{V}$ as a function of Ps − Pao and Pao for N_2, and indicates that, for a given Ps − Pao, flow was greatest at the largest lobe volume (Pao = 15 cmH_2O). Note also the curvilinear shape of the plots. Figure 1*B* demonstrates the effect of the test gas on the $P\dot{V}$ relationship when Pao = 15 cmH_2O. Because the pneumotachograph calibration factor differs for each gas, the position of the curves do not correspond directly to $\dot{V}$. Ne, being approximately three times as viscous as the other gases, requires a larger pressure to generate a similar $\dot{V}$. Figure 1*B* is shown to demonstrate the difference in shape of the four curves. Averaged data for all eight lobes showing $\dot{V}$ in milliliters per second as a function of Pao and Ps − Pao are shown in Fig. 2. As indicated in Fig. 2 and confirmed by the significant interaction terms in the ANOVA, the effect of changing Ps − Pao differed depending on Ptp and the test gas.

A double-log plot of P_n against Re is shown for each Ptp in Fig. 3. For clarity each gas is represented by a different symbol; SF_6, the most dense gas, has the largest Re, and He, the smallest. For every gas, each point represents a different driving pressure, 2 cmH_2O having the largest $\dot{V}$ and, therefore, greatest Re and 0.25 cmH_2O the smallest $\dot{V}$ and smallest Re. Individual plots constructed for each lobe, although exhibiting more scatter, were similar to the composites shown in Fig. 3.

The effect of alterations in Ptp and driving pressure on $\dot{V}$ pattern and the geometry of the $\dot{V}$ conduits can be inferred from Fig. 3. Increasing lobe volume by raising Ptp from 5 to 10 cmH_2O caused a parallel displacement of the curve down and to the right, suggesting that the characteristic dimension of the flow conduits increased and was unaccounted for in the calculation of Re and P_n. A smaller translation resulted when Ptp was raised from 10 to 15 cmH_2O, consistent with the curvilinear nature of the PV curve.

At Ptp = 10 and 15 cmH_2O, data points for all driving pressures appeared to fit a single curve. Below an Re of 100, the curve paralleled a line having a slope of −1, indicating viscosity-dependent, or laminar, flow. At Re >100, slope decreased, indicating the development of a transitional flow pattern. The finding that all data points defined a single curve suggests that when the lobe was inflated to large volumes, small changes in driving pressure did not detectably alter the characteristic dimension of the flow conduits in the collaterally ventilating lung segment. For example, at Pao = 10 cmH_2O, the point representing Ps − Pao = 0.25 cmH_2O for SF_6, and the point representing Ps − Pao = 1.75 cmH_2O for N_2 are identical. A change in dimension between Ps − Pao = 0.25 and 1.75 cmH_2O, which was unaccounted for in the calculation, would have separated these points. Such a separation is seen at Pao = 5 cmH_2O in that the points no longer define a single continuous curve but appear to define three discontinuous linear segments. For similar Re values, points representing larger driving pressures are displaced downward, indicating increased dimensions.

Intact Lobes

Because $\dot{V}$ was not measured at identical driving pressures in intact lobes, data were not averaged or analyzed statistically. Individual curves are therefore presented in Fig. 4 for six dogs successfully studied. Two curves are presented for each dog, with open symbols representing the closed-chest condition and closed symbols representing data collected when the chest was widely opened and 5 cmH_2O positive end-expiratory pressure was being applied at the trachea. As in Fig. 3 each gas is represented by a different symbol, and for every gas, each point represents a different driving pressure. In each case, the curves representing the open-chest condition are beneath those representing the closed-chest condition, suggesting that the characteristic dimension of the flow conduits was larger when the chest was opened, possibly because lung volume was greater. Although lung volume was not measured to confirm this suggestion, when the chest was closed Ptp at end expiration was <5 cmH_2O in *dogs A–D*. All data appear to parallel the line having a slope of −1 indicating laminar flow. For each gas there was no outstanding difference in curve shape between the open-

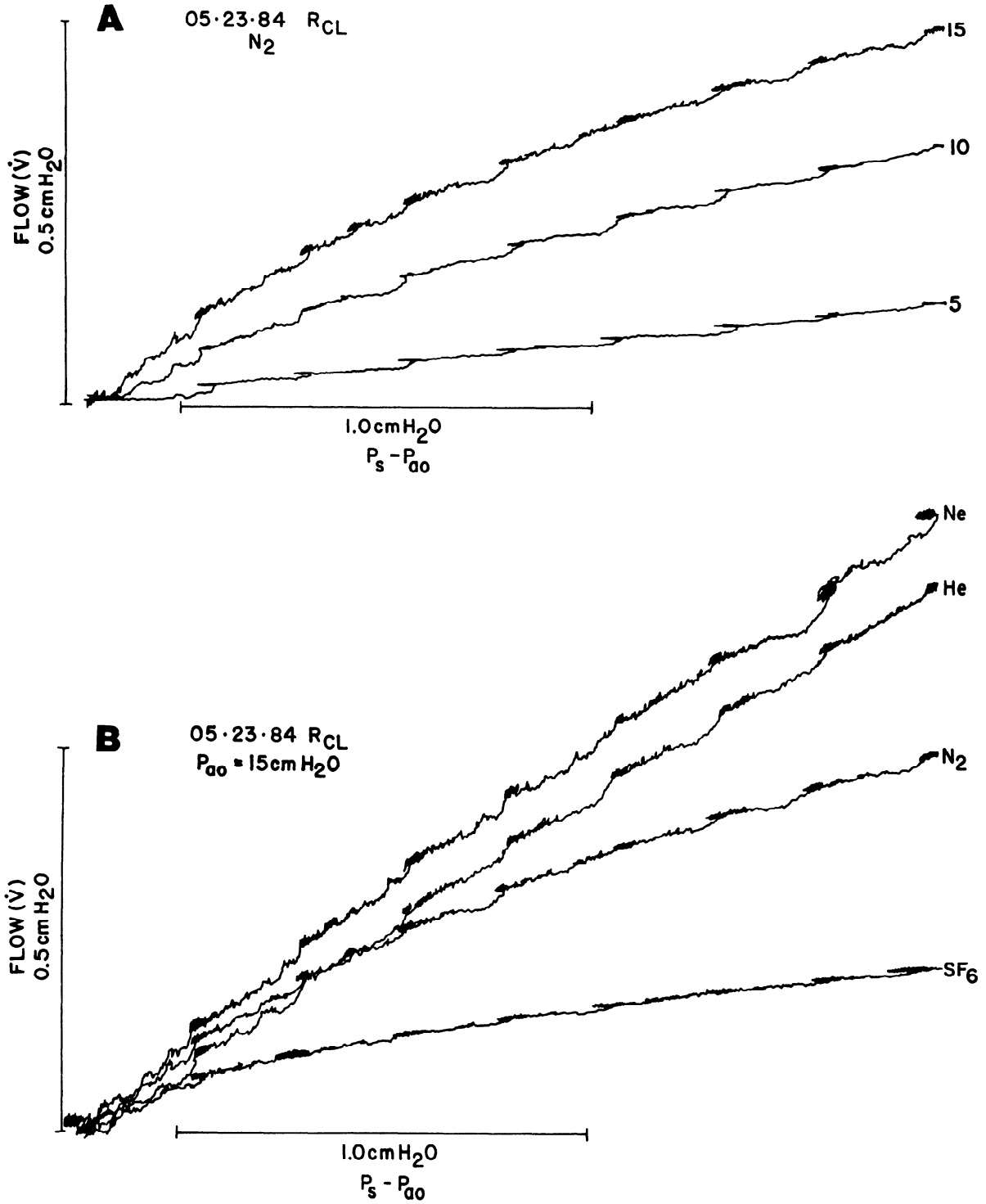

FIG. 1. Representative data from an excised right caudal lobe. *A*: collateral flow of N_2 as a function of driving pressure (Ps − Pao) at Pao = 5, 10, and 15 cmH_2O. *B*: flow of Ne, He, N_2, and SF_6 as a function of Ps − Pao at Pao = 15 cmH_2O. (See text for details.)

and closed-chest condition, although in *dogs B* and *C* and possibly *F* slopes appear reduced at higher Ps − Pao when the chest was closed, suggesting reduced dimensions or transitional flow.

DISCUSSION

This study demonstrated that, in excised lungs, increasing pressure in a sublobar region of lung by 2 cmH_2O did not alter the pattern of gas flow through the lung region or the geometry of the pathways conducting flow unless Ptp was low, and then the effect was small. Similar pressure changes in a sublobar segment in intact lungs at functional residual capacity had no effect. Removing the potential influence of the chest wall by widely opening the chest did not appear to have a consistent effect on the influence of driving pressure on the collateral $P\dot{V}$ relationship in intact lungs.

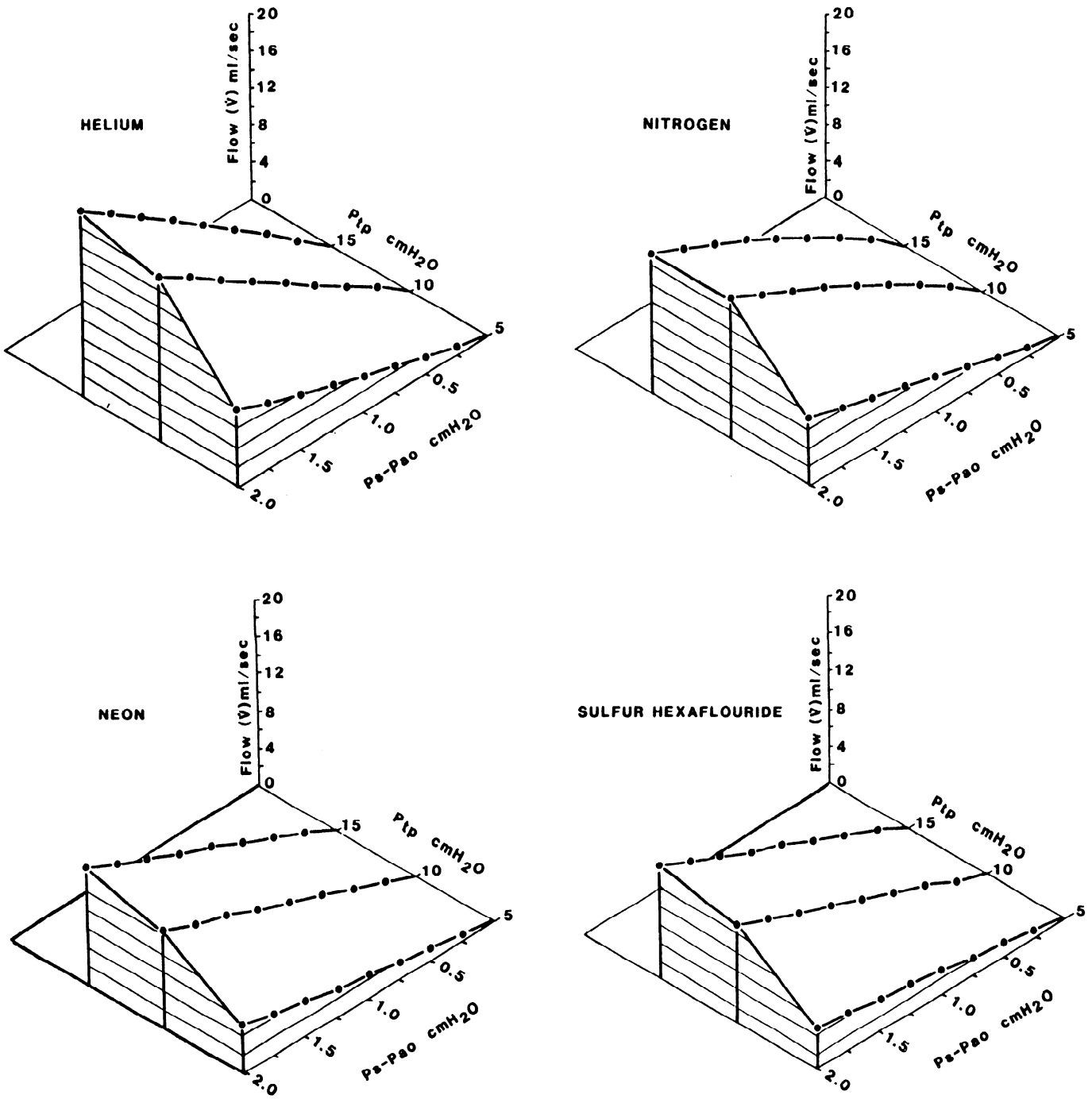

FIG. 2. Average collateral flow ($\dot{V}$) as a function of driving pressure (Ps − Pao) and transpulmonary pressure (Ptp) for Ne, He, N_2, and SF_6 (n = 8 excised caudal lobes).

Technical Considerations

Before discussing the implications of these results, a consideration of potential technical limitations is warranted. Difficulties experienced using excised lobes included the occasional blocking of the pressure port by mucus and the development of gas trapping. Lobes were vacuum degassed and occasionally ventilated with SF_6 to minimize trapping (7). Because trapping increases the parenchymal shear modulus at Ptp <8 cmH_2O, it was anticipated that parenchymal interdependence would be greater in lobes exhibiting trapping (11). Comparison of individual results from lobes with a large increase in minimum volume and those that appeared to collapse more completely showed no difference.

Results were more difficult to obtain in intact lungs. Mucus problems were much more frequent, and segments that could accommodate measurable flow rates for all gases were more difficult to locate. Reflex bronchoconstriction, as evidenced by increasing Ps − Pao at constant flow, frequently occurred initially in response to the test gases, presumably due to local hypocapnia or airway cooling. Atropine and vagal sectioning were used to minimize these effects. At necropsy several segments demonstrated locally trapped gas as evidenced by poor col-

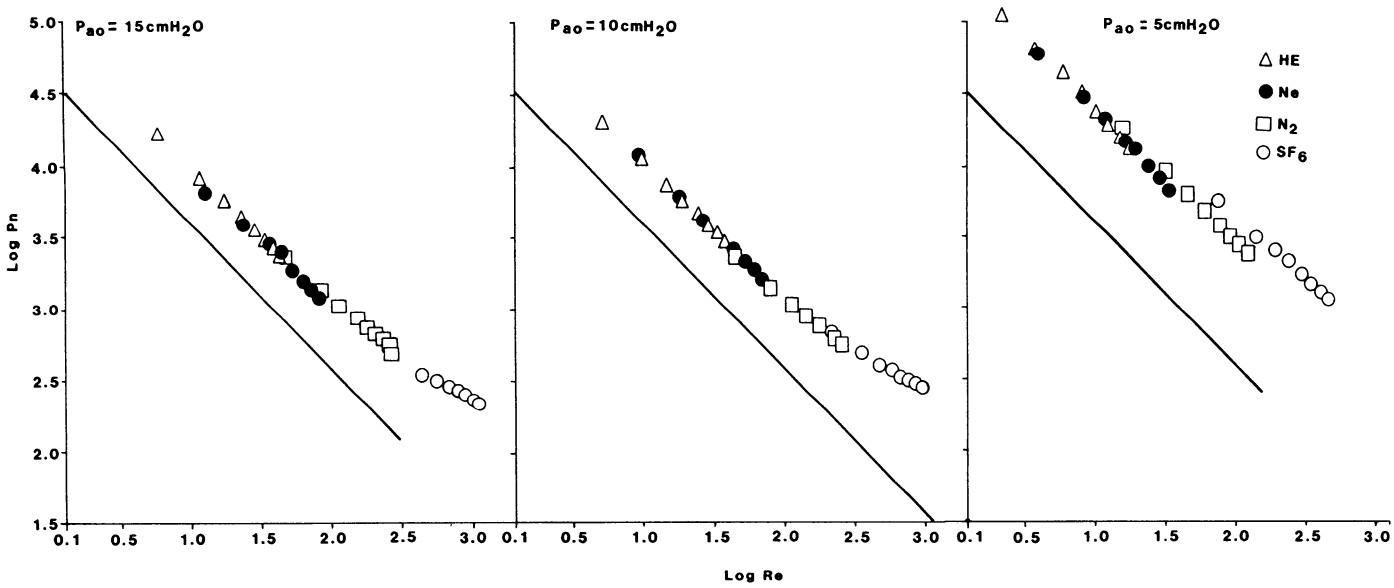

FIG. 3. Double-log plot of normalized pressure (P_n) against Reynolds number (Re) for excised caudal lobes (n = 8). Gases: He (△); Ne (●); N_2 (□); SF_6 (○). Slope of the solid line is −1. (See text for details.)

lapsibility and firmness to the touch. As in the excised lobes, however, the presence or absence of trapping did not visibly alter the results.

Mechanical Interpretation of Data

When gas flows through the wedged catheter, it enters the subtended lung segment and leaves the segment through collateral channels. The fluid mechanics of flow through the distensible branching airways and possibly tortuous collateral channels throughout the segment is clearly complex. The shape of the driving PV̇ curve is a reasonable first estimate for evaluating the mechanics of flow through the segment. A straight-line relationship between pressure and flow would suggest that flow was viscosity dependent, i.e., laminar, and that the dimensions of the flow conduits remained constant as pressure was increased. If flow remained laminar and the flow conduits were deformed by an increase in driving pressure, a nonlinear PV̇ curve would result. Specifically, if the pathways were dilated, flow would increase more rapidly than pressure, resulting in a PV̇ curve that curved toward the flow axis. If increases in pressure in the collaterally ventilating lung segment distorted the pathways and reduced the effective flow area, pressure would increase more rapidly than flow, resulting in a PV̇ curve that curved toward the pressure axis. This analysis is complicated by the potential effect of the development of density-dependent flow in the absence of a change in conduit geometry. Laminar flow is the minimum energy case with flow being directly proportional to pressure ($P \propto \dot{V}^1$). As turbulence develops, the pressure cost to maintain flow increases and is manifest as a PV̇ curve that curves toward the pressure axis ($P \propto \dot{V}^{1+n}$, where $0 < n \leq 1$). Dimensional analysis was used to determine whether the curvilinear PV̇ relationship was due to a change in flow pattern, conduit geometry, or both.

When P_n and Re were computed, it was assumed that the characteristic dimension of the system was independent of changes in airway opening or segment pressure. The validity of the assumption is tested by examining curve placement on the double-log plot of P_n vs. Re. A parallel shift, i.e., translation, of the entire curve indicates that flow pattern did not change but that a change in d was not accounted for. If d was known at each Ptp and P_n and Re calculated accordingly, curves representing different lung volumes should superimpose. We have previously shown that scaling d with the cube root of lung volume superimposes curves for different Ptp levels (18). A similar translation is seen in the present study at the three Ptp levels studied, i.e., in excised lungs and in intact lungs when the chest was opened and 5 cmH_2O positive-end expiratory pressure applied to the trachea.

A change in the slope of the curve, i.e., rotation, indicates that either a dimension change was unaccounted for or that flow pattern changed. A reduction in slope, down to the theoretical limit of 0, suggests the development of more density-dependent flow or reduced dimensions. An increase in slope up to the theoretical limit of −1 suggests the development of more viscosity-dependent flow or increased dimensions.

In excised lungs at Pao = 10 and 15 cmH_2O, data points representing different driving pressures formed a smoothly continuous curve, suggesting that changing driving pressure by increasing pressure in the collaterally ventilating lung segment did not change the characteristic dimension of the flow conduits within it. P_n and Re were therefore computed correctly. If it is assumed that the test gases did not systematically alter the lung segment, the curve defines a single geometry, with flow being strictly a function of gas density and viscosity. Thus the conclusion that the curved PV̇ curves for N_2 and SF_6 at Pao = 10 and 15 cmH_2O are due to the development of a density-dependent flow pattern through the lung segment rather than a change in the geometry of the flow conduits.

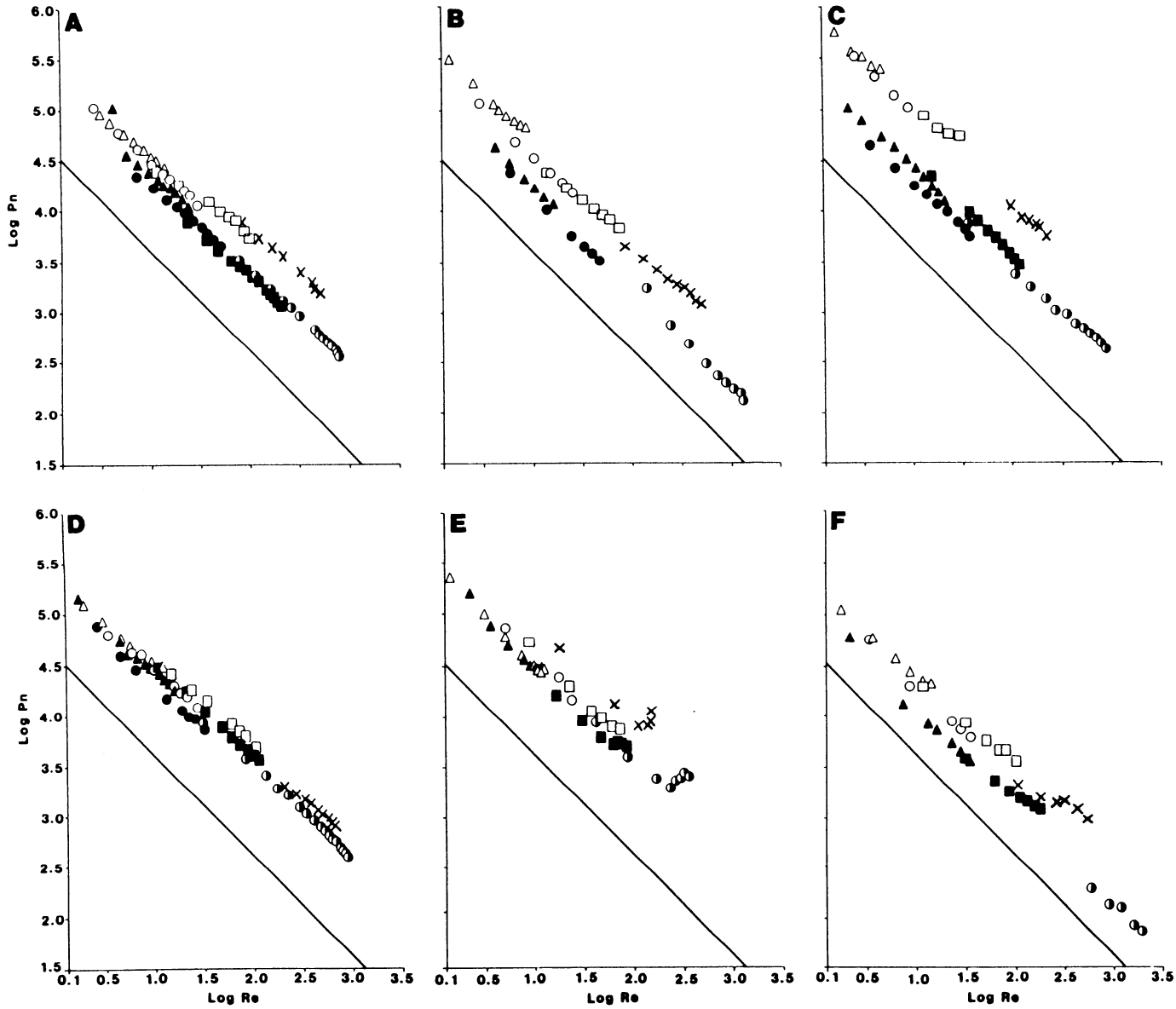

FIG. 4. Individual double-log plots of normalized pressure (P_n) against Reynolds number (Re) for 6 intact caudal lobes. *Open symbols*, closed-chest condition; *closed symbols*, open-chest condition with 5-cmH_2O positive pressure applied to the trachea. Gases: He (△, ▲); Ne (○, ●); N_2 (□, ■); SF_6 (×, ◑).

In contrast, at Pao = 5 cmH_2O the curve formed by all data points is discontinuous. Now the curve segments formed by data points for each gas at identical driving pressures are separated yet roughly parallel. If it is assumed that the dimensions of the flow conduits are a function of driving pressure and are not affected by changing the gas flowing through them, the finding that the portion of the curve representing a driving pressure of 2 cmH_2O is displaced below and to the right of the curve segment representing a driving pressure of 0.25 cmH_2O suggests that when segment pressure is raised to 2 cmH_2O above pressure in the surrounding alveoli, flow conduits within enlarge. This increase in characteristic dimension was not accounted for when P_n and Re were computed. If the change in dimension was known and P_n and Re were recomputed for each driving pressure using the corrected dimension, the curve formed by all points should be smoothly continuous, as for Pao = 10 or 15 cmH_2O. This suggests that although the PV̇ curves appear more linear at Pao = 5 cmH_2O, this linearity does not simply represent viscosity-dependent flow but also a straightening effect due to increased dimensions of the flow pathways as Ps − Pao is increased.

Physiological Interpretation of Data

The dimensions of the pathways conducting flow through the collaterally ventilating lung segment could change passively due to a change in transmural pressure, or change in the elastic recoil of the surrounding parenchyma, or actively due to a change in tone of the airway smooth muscle. Active bronchoconstriction was seen with each gas in the intact lungs when flow was initiated, as evidenced by increased segment pressure at constant

flow. The flow trace stabilized within a few seconds and remained stable at each subsequent segment pressure, indicating no further change in tone. It is therefore unlikely that the apparent change in dimension as driving pressure changed was due to bronchoconstriction. Passive changes in transmural pressure could occur by alterations in intrabronchial pressure, peribronchial pressure, or segment volume. Alternatively, additional collateral pathways could be recruited or derecruited, changing the total effective cross-sectional area for flow. The effect of a change in transmural pressure on any given airway will be determined by its deformability and the deformability of its surroundings.

In excised lobes, bronchial diameter increases asymptotically as Ptp increases, reaching 90% of maximum diameter (diam at Ptp = 25 cmH_2O) at a Ptp of about 10 cmH_2O (11). When lobe volume is held constant and pressure reduced in a lobar bronchus after obstructing the tertiary bronchi with beads, the slope of the calculated diameter-transmural pressure curve is greatest at Ptp = 5 cmH_2O and smallest at Ptp = 30 cmH_2O, indicating bronchial deformability during a nonuniform deformation to be lung volume dependent (16). At low lung volumes bronchial diameter is more sensitive to changes in transmural pressure. The results of the present study can be interpreted in conjunction with these observations and the knowledge that parenchymal deformability is inversely related to lung volume (11). In the excised lobes inflated to large lobe volumes (Pao = 10 and 15 cmH_2O), small increases in segment pressure increased flow but did not appear to change the characteristic dimension of the pathways conducting flow, presumably because these pathways were nearly maximally dilated and the stiff parenchyma surrounding them resisted deformation. In the excised lobes at Pao = 5 cmH_2O, a lobe volume where both airways and parenchyma are more deformable, the curve formed by all gases was discontinuous, suggesting that increasing segment pressure dilated the pathways conducting flow or recruited additional pathways.

Conclusions based on the intact lung data are more tenuous because of the inability to average or statistically analyze the data. Roughly continuous parallel lines having slopes approximately equal to −1 were formed in the open- and closed-chest conditions, suggesting no effect of either segment pressure or chest wall on the collateral $P\dot{V}$ relationship. In two cases, the intact curve appeared discontinuous with reduced slopes for each gas suggesting reduced dimensions, but no firm conclusions may be drawn. The separation of the open- and closed-chest curves suggests that the pathways conducting flow were larger in the open-chest condition, presumably because lung volume was larger.

It is unclear why raising Ps − Pao apparently altered the dimensions of the conduits in excised but not intact lungs at a Ptp of 5 cmH_2O. There are several likely causes. First, it is possible that the scatter in the data from the intact lungs obscures the effect. Second, it is possible that simply opening the chest did not remove the interaction between the lobe containing the collaterally ventilating segment and the surrounding structures such as diaphragm, neighboring lobes, and fluid in the costodiaphragmatic recess. Third, the intact pulmonary circulation may have altered parenchymal distensibility, although raising pulmonary venous pressure from 5 to 30 Torr in excised perfused lungs has no effect on the pulmonary PV curve at comparable lung volumes (2, 3). Finally, the bronchoconstriction apparent in the intact lobes may have altered the pressure-diameter behavior of the airways, making them stiffer (11), and therefore not altered by a 2-cmH_2O change in intrabronchial pressure. A more complete study of intact lungs is probably warranted.

It is difficult to compare the results of the present study with previous investigations into the effect of segment pressure on collateral flow because of the methods used. Prior conclusions have been based on the interpretation of changes in collateral resistance (Ps − Pao/$\dot{V}$) that fails to separate the possible effect of an alteration in flow pattern from a true geometrical change. This study suggests that raising Ps could increase dimensions which would reduce resistance and increase flow which could increase resistance if flow was not laminar. This potential dilemma highlights the necessity of knowing flow pattern and potentially explains results in excised lungs, demonstrating that increasing Ps had no effect on collateral resistance when Pao = 2 cmH_2O but increased resistance when Pao = 5 cmH_2O (4).

Finally, it must be emphasized that, unlike previous studies, the pressure in the collaterally ventilating segment was changed by only 2 cmH_2O. It is possible that larger increases in pressure could have a different effect by causing distortion at the parenchymal interface between the segment and surrounding parenchyma through which collateral channels must pass (4).

Technical considerations aside, it is tempting to speculate on what these results might indicate regarding canine physiology. If it is assumed that the N_2 data in excised caudal lobes can be extrapolated to inspiratory airflow in intact lungs, several suggestions can be made. If inspiratory time is ~1.3 s and the volume of the collaterally ventilating segment is 50 ml, then an inspiratory effort of only 2 cmH_2O would deliver a regional tidal volume of 10 ml or 20% of regional volume to a region distal to an obstructed airway. If the primary route for collateral ventilation were airways rather than pores, it is likely that the gas reaching the segment would be fresh gas. The flows occurring with even smaller pressure differences suggest that regional differences in alveolar pressure caused by time constant inequalities or even cardiogenic pressure oscillations might promote interregional mixing which could contribute to the efficiency of gas exchange by reducing the diffusion distance for respiratory gases.

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